Introduction

I've always had a weight problem. I would rarely have been considered fat, but I was always trying to lose weight. When I was eight years old, I wanted to get thinner so I could look sharp in my Brooklyn Dodgers uniform to impress Barbara Levy, who was the most beautiful girl in the world as far as I was concerned. I don't recall having any great success, and it was only fairly recently that I found out that Barbara Levy is now Barbara Boxer, former senator from California. In any case, I always knew that starch made me fat—oddly, I was less afraid of sugar because I mistakenly believed that there wasn't that much in Coca-Cola and the other sodas I drank. I grew up with what is usually called a poor self-image, and as the old joke goes, inside of every Botero is a Giacometti trying to get out.

I knew from early on that it was important to cut out starch and obvious solid sugar, and I made other observations about diet—for example, that cold cereal for breakfast made me slightly sick. It's difficult for me to remember exactly what I did eat in the morning. At least some of the time it was bacon and eggs, which, in those days, was just one of the things that people ate. Nobody recoiled in horror at bacon. The only dietary advice at the time was to eat from the different food groups, which were represented by a pie chart with unique symbols in each slice. The bottle of milk was one that stuck in my mind. I felt early on that it was not interesting, and I was sure that I didn't need an "expert" to tell me what to eat. When the USDA food pyramid was introduced, I knew it was a crock and I assumed that others did, too. My principles were simple: If you have a weight problem, bread will make you fat, and if you don't have a weight problem, why do you need the USDA? I thought everybody was in agreement on that, but obviously that wasn't the case. I'm not sure why people went along with all the "expert" advice. After all, everybody has a great deal of experience with food. We all do three experiments in "nutritional science" each day.

People's compliance with dietary standards probably has to do with the history of medicine. Among the turning points in that history was the discovery of vitamins. Unlike poisons and microorganisms, vitamins were stuff that you had to take if you didn't want to get sick. Another inflection point was the identification of cigarette smoke as a causal agent in lung disease. In that case, even though there was a toxic agent, the associations were subtle and one needed statistics or other expert insights to see the connection. This subtlety might have given people the idea that there were experts who could see harm where they couldn't.

In my youth, I simply ignored the "expert" advice. I thought that I knew what to eat (I was mostly right), and I saw obesity as a personal rather than professional question. Decades later, when I began teaching metabolism, I had to confront the interaction between science and nutrition. It proved to be more difficult than I would have guessed.

This book is the story of my encounter with the world of nutrition, a story of the science of biochemistry and metabolism—how you process the food that you eat. It is about the application of science to daily life, which is what I like about the subject. If you know a little chemistry, you can appreciate the way that human evolution has reached into the mixing pot of chemical reactions to obtain energy from the environment, and even if you don't know chemistry, you can see the beauty in the life machine.

But there is another side to the story. In the contentious and continually changing stories of nutrition in the media, I encountered a discouraging example of the limitations of human behavior in facing truth and preventing harm. The story of nutrition has proved to be an almost unbelievable tale of poor and irresponsible science within the medical community, one of the most respected parts of our society.

However hard it is for scientists to distrust experts, it is even harder for the general population. I was astounded when I saw a question on an online diabetes site that said, "My morning oatmeal spikes my blood glucose. How much carbohydrate should I have?" People with diabetes cannot adequately metabolize dietary carbohydrate (starch and sugar) so it seemed like an easy question. The answer from the experts, however, was waffling and tedious, and it didn't include the obvious advice: "Limit your oatmeal consumption to a level that doesn't spike your blood glucose."

Chemistry

When I was eight years old, my father taught me about atoms. I have one of those memories that might or might not be accurate: I am sitting in my father's car, and he is telling me that the whole world is made of atoms in the same way that the apartment building across the street is made of bricks. Whether or not the scene really took place, it was a major influence, and chemistry has long been a defining feature of my life. (Other vivid memories of my early life in Brooklyn—being at Ebbetts Field and seeing Jackie Robinson hit an inside-the-ballpark home run—turned out not to be true. He had hit only one, in 1948, before I had ever seen a live game).

The crux of atomic theory, the thing that captures everybody's imagination when they are first exposed to it, is that it is a global and absolute theory—it explains everything that has been done in the laboratory, the kitchen, or anywhere else. Various fields of chemistry get at that same sense of universal understanding with differing degrees of intellectual rigor, but eventually I recognized that biochemistry was a good place to be for a young person who didn't know what career he wanted to end up with. You can do drug design or theoretical chemistry or animal behavior or nutrition and still call yourself a biochemist.

Teaching Nutrition

I have worked in a number of fields in biochemistry, but it was teaching metabolism to medical students at SUNY Downstate Medical Center that led to my professional interest in nutrition. Metabolism is the study of the way food is processed and of the biochemical reactions that control life functions. It is a fairly complicated subject—those parts that we understand at all. Because there are so many individual biochemical reactions, students tend to see the subject in the same way that somebody once described the study of history: just one damn thing after another. There are general principles and big concepts, of course, but you do have to know the details. When I began teaching metabolism, I used the low-carbohydrate diet—at the time primarily a weight-loss diet—as a central element in my teaching. Control of blood glucose and insulin, the hormone whose release is controlled by glucose, is central to many different processes in

biochemistry. In the complicated network of biochemical reactions, insulin stands out as a major point of regulation. The ups and downs of insulin are what we try to control through the use of dietary carbohydrate restriction as a therapeutic method. So, low-carbohydrate diets provided some unifying theme in teaching. I still teach metabolism in this way, though I now emphasize diabetes where impaired ability to handle dietary carbohydrate is the salient feature. The low-carbohydrate diet and its more thorough form, the ketogenic diet, are popular—periodically very popular—and while they remain controversial, the number of adherents, and possibly the desperation of the detractors, suggests that low-carbohydrate must inevitably be accepted as I will describe it: the "default" diet for diabetes (the one to try first) and the best diet for weight loss for many people. It is likely that its current popularity can't be turned back. Myself and others who use this teaching method have published papers about how understanding the real-world benefits of low-carbohydrate—getting control of your health and regulating your weight—can help you learn chemistry.[1]

Around 2000, one of our second-career medical students who had been a dietitian suggested that we include formal nutrition in our biochemistry course, and she provided subject matter from standard nutritional practice. I cannot really describe what it was about—probably, even at the time, it was so vacuous that I couldn't retain it in my memory. In any case, I objected because whatever it was, it wasn't biochemistry. The way I saw it, criticizing how lectures are given is like complaining about how the dishes are done: Everybody sees an immediate solution. Despite my protests, I wound up having to give formal lectures in nutrition, and I really didn't know the literature. I had long ago found that carbohydrate restriction was best for me, and while low-carbohydrate diets provided me with a good framework for teaching metabolism, applied therapies do not always have a close relation to theories, so teaching nutrition required a certain amount of background study on my part.

My first lectures on nutrition were neutral. I simply tried to cover the basic aspects of low-carbohydrate and low-fat diets—the two main choices, really—presenting the pros and cons of each approach in a simple way. Low-fat diets are not based substantially on biochemical mechanisms; instead, they follow from observed correlations between cardiovascular disease and the presence of cholesterol or other lipids in the blood.

More recently, low-fat has morphed into a prescription for obesity, and proponents have started emphasizing the point that fat has more calories per gram than other things, peddling the idea that the more calories, the greater the effect on body weight—the ill-conceived idea that "you are what you eat," which hangs over everything. While I could explain at that time how metabolism, and specifically the role of the hormone insulin, accounted for the benefits of a low-carb diet, I could not provide a well-organized review of the relevant studies in the medical literature. So, my initial lectures were rather simple and straightforward while I tried to get a grip on the scientific literature.

As I dug into that literature, however, it didn't take long to see that something was terribly wrong. In simply trying to grasp the facts, I had stepped into a world of bad science, self-deception, and a scandal equal to any in the history of medicine.

The Nurses' Health Study

Science is very specialized. Although I had been doing research on blood coagulation, which is related to cardiovascular disease (CVD), I did not pay much attention to the diet–heart hypothesis—the idea that fat and cholesterol in the diet raise blood cholesterol, which, in turn, leads to CVD. I was suspicious of such a theory, though, because biology tends to run on hormones and enzymes—that is to say, on control mechanisms rather than on mass action (the principle that chemical processes are determined by how much reactants are put into them). The grand principle in biochemistry is that there is hardly anything that is not connected with feedback. If you try to lower your dietary cholesterol, for example, your liver will respond by making more. Simply adding more or less is not guaranteed to produce much change at all, once feedback is taken into account. I was therefore skeptical, if not well-informed.

Whatever my misgivings about the diet–heart hypothesis, I didn't question it very deeply at first. However, when I went back to the original literature to find the evidence supporting low-fat recommendations, as I had to do in preparation for my lectures, it was a rude awakening. My assumption that there was at least a grain of truth in the diet–heart hypothesis turned out to be overly optimistic. If the hypothesis is not a

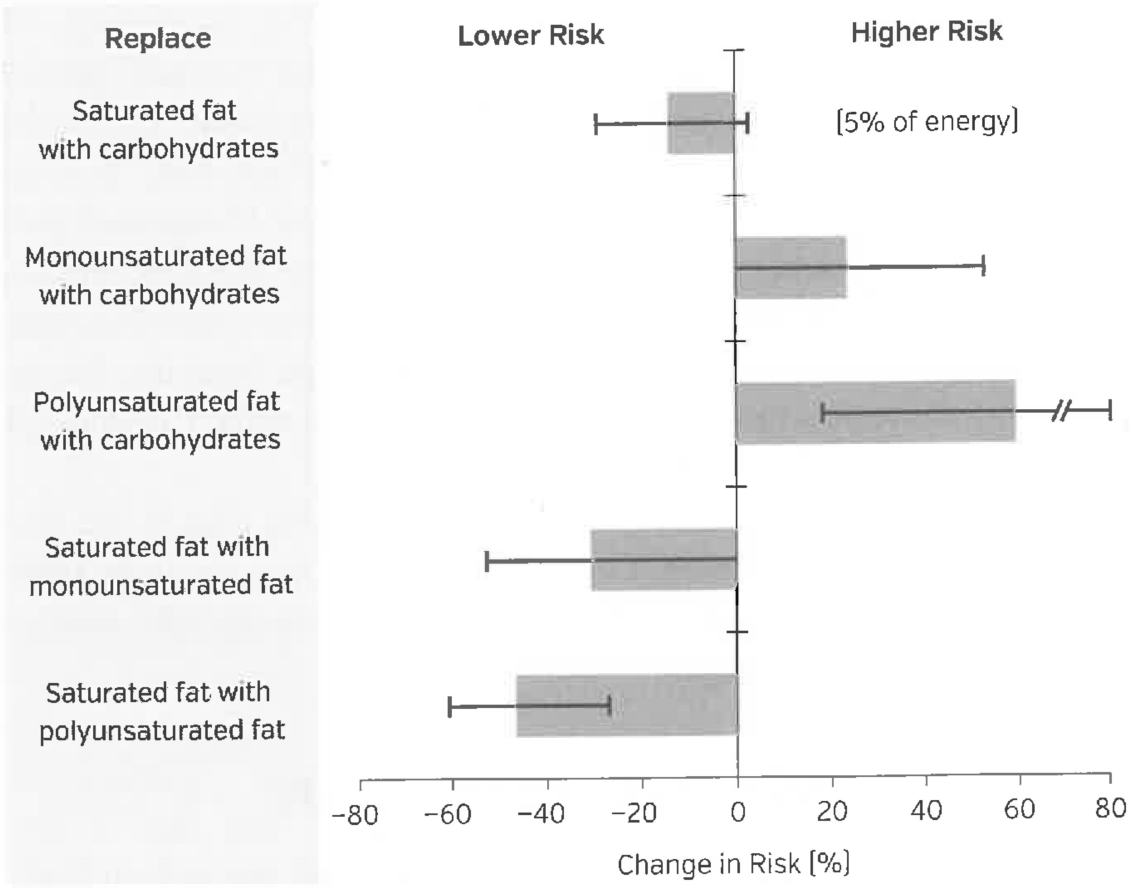

Figure 0.1. Estimated changes in risk of coronary heart disease associated with isocaloric substitutions [error bars show 95 percent confidence interval]. Adapted from F. B. Hu et al., "Dietary Fat Intake and the Risk of Coronary Heart Disease in Women," *New England Journal of Medicine* 337, no. 21 (1997): 1491–1499.

total sham, it is pretty close. One of the first papers that I came across in my literature survey was a report from the Nurses' Health Study (NHS). Centered at Harvard, the NHS is one of the largest epidemiological studies with more than one hundred thousand participants. It has produced a large number of studies on nutrition and other aspects of lifestyle.

Walter Willett, head of the NHS and follow-up studies, and his associate Frank Hu examined the association of different kinds of fat, as well as carbohydrate, with the risk of CVD.[2] I found the result astounding. Figure 0.1, redrawn from their paper, shows the effects of substituting one type of fat for another, and of substituting carbohydrate for fat. Replacing saturated fat with either polyunsaturated fat (vegetable oils) or monounsaturated fat (olive or canola oil) reduced risk substantially. That's what the nutritional community had been saying, so I saw no surprise there.

However, when the polyunsaturated fat was replaced with carbohydrate, Hu et al. found an average 60 percent increase in risk. What? Carbohydrate is worse than fat for cardiovascular risk? That's not how it was supposed to be. What about saturated fat? Surely that's a bad guy. Replacing saturated fat with carbohydrate did provide some benefit according to the figure, at least on average, but there is more to the story. In this kind of figure, the error bars (horizontal lines) show the spread of individual values, which was quite large in this case. In other words, even though there was an *average* improvement from replacing saturated fat with monounsaturated fat—their main selling point—some subjects experienced greater benefit than the average, and some much less benefit than the average. When saturated fat was replaced by carbohydrate, some of the study's subjects were, in fact, going in the opposite direction—that is, they were at greater risk for CVD, which contradicted the supposed benefit of reducing fat. It wasn't just a few subjects, either. The breakdown was about 60:40: 60 percent of subjects experienced reduced risk of CVD by replacing saturated fat with carbohydrate, and 40 percent experienced greater risk. It gets worse. Without getting too caught up in the statistical details, the rule is that if the (horizontal) error bar crosses the zero line, then there is no significant effect of the substitution. So, based on the study's findings, substituting carbohydrate in place of saturated fat is at best neutral, or more precisely, it is as likely to increase risk as it is to lower it. The same is true of substituting carbohydrate for monounsaturated fat.

Looking at figure 0.1, it is hard to see a risk of fat, but hasn't risk from fat been the message all along? Certainly the idea that carbohydrate is a risk is not found in the media or the pronouncements of health agencies. And then there is the authors' summary of the paper:

> Our data provide evidence in support of the hypothesis that a higher dietary intake of saturated fat . . . is associated with an increased risk of coronary disease, whereas a higher intake of monounsaturated and polyunsaturated fats is associated with reduced risk. These findings reinforce evidence from metabolic studies that replacing saturated fat . . . with un-hydrogenated monounsaturated and polyunsaturated fats favorably alters the lipid profile, but that reducing overall fat intake has little effect.

This conclusion is not accurate. It's at best misleading, and at worst outright deceptive. The measured risk of saturated fat intake was dependent on what it was replaced with, yet there is no mention of carbohydrate as a replacement. The most striking thing to me was that if you looked at the risk from carbohydrate in comparison to the risk from saturated fat—that is, the risk of substituting one for the other—there was no difference. Even worse, substituting carbohydrate for other fats increased risk. How could this be? Fat out. Carbohydrate in. Wasn't that the clear recommendation for improved health from just about every health agency and expert? Yet the data said it didn't matter. Was it dishonest not to make this clear in the discussion section of the paper? At best, it was an error of omission. The authors from the Harvard School of Public Health were, and still are, the more modest among those vilifying fat, insisting that it is only the type of fat that we need worry about. Most recently, the American Heart Association (AHA) has come around to the same point of view as if they had just discovered it. I was probably not alone, but I began using the term *lipophobes* long ago for proponents of low-fat. It's a wiseguy term, and since I was still something of an inside player in the nutrition world, I was reluctant to put it into print until Michael Pollan started using it without any sense of irony.[3] (I started saying "...as Michael Pollan calls them.")

By the time I read the NHS paper, my professional involvement in the field of nutrition was cemented. I did not, however, adequately attend to the sense of being sucked into a whirlpool from which it would be hard to escape. The data supporting low-carbohydrate were there for everyone to see, I thought, even if the authors had chosen to downplay the strongest result.

A trip to the supermarket today demonstrates that the results from the Nurses' Health Study had little effect. The low-fat story is still with us. More striking is that two meta-analyses (averages of many studies) came to the same conclusion regarding the ineffectiveness of replacing fat with carbohydrate. Siri-Tarino et al. concluded that "there are few epidemiologic or clinical trial data to support a benefit of replacing saturated fat with carbohydrate," and in March of 2014, yet another meta-analysis found similar results.[4] What turned out to be most remarkable about all of these studies was that they presented a reanalysis of studies that had found no effect of saturated fat to begin with. One has to ask why the results were not accepted when first published. Some of the included studies are

twenty years old. How is it possible that, in the most scientific period in history, our society runs on incorrect scientific information? That's one of the questions that I will try to answer in this book—or at least describe, as I'm not sure that there is a clear-cut answer. Looking ahead, I will introduce the revolutionary idea that, except in cases of well-defined genetic abnormalities, there is no predictable effect of diet on heart disease based on the current research. It is a hypothesis, and we might know more as we understand the genetics, but no effect is certainly more plausible than the diet–heart hypothesis, which remains only a conjecture without experimental support. This lack of effect will be one of the themes in this book and one of the battlegrounds as the crisis in nutrition plays out.

About This Book: Who It's for and Why I Wrote It

Food and chemistry have been two of the largest influences in my life. The beauty of biochemistry is that it relates the movement of electrons to what's on our plates—and this is a connection I thought I could explain. I like writing about biochemistry. It allows us to see how things fit together, but it also exposes the things we don't know—the things that evoke within us curiosity, the defining feature of the scientific life. If you want to indulge that curiosity, you are the person I had in mind when I started the book.

This is a book for scientists. Not specifically for people with atomic-force microscopes in their labs, but for those who want to look at nutrition from a scientific point of view. Science is less about sophisticated measurements than it is about basic honesty. It is true that scientific fields can be very mathematical or intellectually rigorous, but all sciences, even those as complex as quantum mechanics, are tied to logic and common sense, and are frequently directly accessible to lay audiences. Part of the game, most researchers understand, is to make the results easy to understand. Einstein is widely quoted as having said that we want to make it simple but not too simple. Modern medicine, despite its reliance on technology, explicitly accepts an obligation to explain things logically to the patient. It doesn't always fulfill this obligation well, but the goal remains nonetheless. In this book I will try to fill some of the gaps and define words, but for the tough spots, you will have to read like a scientist. How do we read? We're all specialists so most of us can't read technical articles, even those in our own

fields, without some bumps. Skip over the bumps and see if you can get the big picture. You can always come back to them later, and many of the details are just a Google away.

If you write a book about biochemistry, it's about chemistry, but if you write a book about nutrition, it's about everything. Not every chapter in this book is for everybody. I have tried to provide a continuous, easy-to-follow thread, but different subjects require different kinds of discussion, and some of these discussions are necessarily technical. You can skip them, but I do suggest giving them a shot.

Although this is primarily a book about the science of nutrition, you can't escape the sociology and politics of medicine. Establishment medical journals, private organizations, and government health agencies have insisted on low-fat, low-calorie dogma despite the scientific evidence to the contrary. This politically motivated breakdown in scientific practice is deeply discouraging to me and was an additional motivation for my writing this book. The corruption of science goes beyond principle, too—what's at stake is the health of patients.

The breakthrough in understanding metabolism that underlies much of this book comes from the realization that many superficially unrelated pathologic or disease states and associated conditions are intimately connected at the physiological and biological level. Equally important, control of these states rests in a major way with diet. Diabetes, obesity, cardiovascular disease, states of hypertension, and numerous other physiologic states are all tied together. The promise is that, examined together, they might provide a global theory of metabolism and with it a common cure.

A major focus of this book is the concept of metabolic syndrome, which is a collection of clinical markers—including overweight, high blood pressure, and the so-called atherogenic dyslipidemia (the lipid markers that are assumed to contribute to cardiovascular disease)—that together and in combination indicate risk of disease. The identification of metabolic syndrome constitutes, in my view, a great intellectual insight. That the common effector is likely the hormone insulin points to the importance of controlling dietary carbohydrate, the major stimulus for insulin secretion.

The resistance of the medical profession to dietary carbohydrate restriction in the treatment of metabolic syndrome, and even more obviously,

in the treatment of diabetes, I find incomprehensible. Everybody knows somebody with diabetes. Echoes of the early days in Brooklyn made it very upsetting to see pictures of Jackie Robinson taken shortly before his death from diabetes complications at age fifty-two. Because it is progressive, the disease is an underappreciated source of suffering. Clinicians will tell you that it is like cancer in its devastating effects. Diabetes is the major cause of amputations after accidents and the major cause of acquired blindness. That is a motivation for writing this book and why you might find it important.

This resistance is a scandal at the level of Ignaz Semmelweis, an early-nineteenth-century Viennese physician. To reduce the incidence of puerperal fever (infection after childbirth), Semmelweis suggested that physicians wash their hands after performing autopsies and before delivering babies. They refused; it was too much trouble. But that was the nineteenth century, before the germ theory was established, and that's some kind of excuse. It's hard to know how we will look back on the actions of the American Diabetes Association (ADA), who believe that for people with diabetes, "Sucrose-containing foods can be substituted for other carbohydrates in the meal plan or, if added to the meal plan, covered with insulin or other glucose-lowering medications."[5]

The most difficult part of writing this book was trying to understand—if such a feat is even possible—how the whole field of medical nutrition could be wrong. Way wrong. Totally off the mark. As misguided as the alchemists' pursuit of the creation of gold. This disconnect from true science is not only bizarre; it is a source of real harm to patients. The phenomenon is particularly hard to explain because the widespread respect for the medical professionals is based on real accomplishment and expertise, and it is hard to see why they would go so wrong in nutrition. For me, having precedents makes it easier to grasp, if not completely comprehensible. Here's one example of self-deception and refusal to accept evidence that I keep in mind. The following is a passage from Abraham Rabinovitch's writing on the Israel Defense Forces and intelligence in the days before the Yom Kippur War (1973):

> The intelligence chiefs believed they knew a deeper truth . . . that rendered irrelevant all the cries of alarm going up around them. Zeira and his chief aides were to demonstrate the ability of even brilliant men to adhere to an idée fixe in the face of mountains

> of contrary evidence. . . . They clung to their view even though the Egyptian deceptions were contradicted by evidence of war preparations that AMAN's [military intelligence] own departments were daily gathering. . . . But the deception succeeded beyond even Egypt's expectations because it triggered within Israel's intelligence arm and senior command a monumental capacity for self-deception.[6]

The Israelis could have lost it all. They could have lost the whole country due to their refusal to accept the evidence. They were largely saved, however, by a couple of field commanders who were wild and crazy guys—most notably Ariel Sharon, who attacked an Egyptian emplacement by disobeying orders not to cross the Nile. Audacity and the refusal to follow orders might be what save nutrition as well.

Finally, this book is for the person (and those for whom she spoke) who posted on my blog wondering how she could determine which nutritional studies are flawed and which are not, especially at a time when we are inundated with so many conflicting recommendations. "Where are the true studies that are NOT flawed," she wrote, "and how do I differentiate?"

She was right to be suspicious. It is not always easy. There are so many nutrition papers that try to snow you with technical detail, and those are in fact the ones to be most suspicious of. Scientific papers will necessarily have technical components, but researchers shouldn't be making their results more difficult to understand than they need to be—and some of the papers are simply not true. Most researchers know that if you make up the data on a federally funded grant, you can go to jail, but when it comes to interpreting the data, they can say just about any damned thing. In this book, I explain how to interpret nutrition papers. In particular, I explain what the statistics mean, how they can be misused, and how to navigate the literature as someone who doesn't necessarily have a background in statistics.

The Second Low-Carbohydrate Revolution

The killer app, so to speak, of the low-carbohydrate diet is still the treatment of diabetes. Intuitively obvious, proved in many experimental trials, and widely used anecdotally and clinically, there are virtually no

contraindications. Resistance to its use appears to be spurred on entirely by pressure from political organizations, primarily the American Diabetes Association (ADA), which, looking for a way to save face, still refuses to endorse low-carbohydrate strategies. Many identify the influence, direct or indirect, of drug companies and food companies as a culprit as well. Whatever the motivation, not encouraging physicians to at least offer carbohydrate restriction is seen by many who have had success with the approach as "criminal." The latest guidelines from the ADA emphasize "individualization," presumably as a way of softening their previous opposition to low-carbohydrate. The word *individualization* is used twenty-one times in their position paper,[7] but the actual principles to be applied for each individual are not stated. The foolishness of not explicitly restricting carbohydrate for people with a disease whose most salient manifestation is an inability to adequately metabolize carbohydrate is astounding. Individualization, in my view, can best be described as a cop-out.

Despite the resistance to low-carbohydrate, we have at the same time a constant flow of blog posts and books that show the low-fat diet–heart hypothesis for the intellectual and clinical disaster that it really is. The most recent and most complete, a book called *The Big Fat Surprise*,[8] is surprising in its description of the depths of self-delusion, if not dishonesty, in keeping low-fat alive. While the pace of criticism is increasing, these exposés document that the diet–heart hypothesis has been debunked since its inception.

If you step back and look at the data, the concerns, the voices on Huffington Post, or the numerous blogs belonging to dietitians, it shines through that the easiest way to lose weight is the low-carbohydrate diet. The concerns, voiced for forty years, have never been effectively substantiated, and the real-world tests of carbohydrate restriction come out in its favor. There are now dozens of successful implementations, though the Atkins diet is still the best known, having attained a somewhat generic status, like Kleenex.

Metabolic Syndrome: The Big Pitch

There is almost nothing in biology that is not connected with feedback. This idea is fundamental yet widely ignored. Reducing dietary intake of cholesterol will have limited effect because of compensatory synthesis.

Likewise, if you stop eating carbohydrate, your body will respond by synthesizing glucose and making other fuels available. This grand idea puts severe limitations on what you can do (as in the case of cholesterol or, looking ahead, trying to starve tumors by reducing glucose), but it also points to some opportunities. When you consume carbohydrate, the hormone insulin turns off the feedback system in the liver that produces glucose from glycogen or gluconeogenesis. Understanding that diabetes represents a breakdown in this feedback system—that the liver of a person with type 2 diabetes will not respond to insulin (insulin resistance)—makes clear why you should not add more insulin. Nonetheless, the compensatory feedback response to many drugs and foods does call for caution in jumping to conclusions.

The key point is that there is a global effect of the hormone insulin. We can get very far simply by regulating this hormone. The role of feedback is part of the picture, but the effects of manipulating insulin can be highly predictable, which is the main theme of this book. Always in the background of this discussion is metabolic syndrome (MetS). Metabolic syndrome is rooted in the observation, generally credited to Gerald Reaven, an endocrinologist who died recently, that a collection of seemingly different physiologic effects—overweight, high blood pressure, high blood glucose, high insulin, and the collection of blood lipid markers referred to as atherogenic dyslipidemia (high triglycerides, low HDL)—are all tied together by a common causal thread: disruption in the metabolic response to insulin.[9] The physiologic markers of MetS predict progression to the associated disease states (obesity, diabetes, hypertension, and cardiovascular disease), all of which respond to dietary carbohydrate restriction. That is the big pitch. This observation confirms that it really is a syndrome (since it has a common underlying cause) and simultaneously points us to the most effective treatment. No dietary approach is better than low-carbohydrate and no drug will target all of the markers together.

There are, in fact, critics of MetS who question the practical significance of the syndrome. What they're really suggesting is that the effects have to be treated with a collection of drugs: drugs for diabetes, drugs for heart disease, drugs for high blood pressure. A low-carbohydrate diet, which is already widely accepted as effective for weight loss, is likely *the* strategy

to treat the different facets of MetS without using this cocktail of drugs. Acceptance of such a notion is the goal of the revolution.

Oddly enough, the bright light on the horizon is the ketogenic diet for cancer. I say "oddly" because carbohydrate restriction for diabetes is already a slam-dunk, and should have been the crystallizing point for change. Of course, as I've already discussed, the resistance to low-carbohydrate for people with diabetes has been extensive. Somehow treatment of cancer is not encountering the same obstinacy, despite limited research on the subject. In chapter 19 I describe work by my colleague Dr. Eugene J. Fine, targeting insulin in the treatment of cancer. I see this study, despite its small size, as a reason for hope and a sign of future progress.[10] If it turns out that we learn to treat diabetes by learning to treat cancer, it would not be the strangest thing that ever happened in science.

How to Approach This Book

If we had ham, we could have ham and eggs, if we had eggs.

—Old American idiom

This book represents the view of a practicing biochemist, and as such, it approaches nutrition as applied biochemistry. Biochemistry is not all there is to nutrition, but it represents a more scientific and logical perspective than "you are what you eat"; it tells us instead that we are what our metabolism *does* with what we eat. I will explain why low-carbohydrate diets are the default diet (the one to try first) for diabetes and metabolic syndrome and why you need to understand this idea even if you are not suffering from either condition. To be clear, I am not an advocate of anything. In the end, you have to be your own nutritionist. My job is to give you some tools for sorting out the army of nutritional "experts" out there and the studies they produce.

I am not an expert on politics, but, as in the aphorism most often attributed to John Adams (I think he stole it from the ancients), I study politics so that my children can study biochemistry and nutrition. It's all tied together. The science is not divorced from the politics. The Framingham Study,[11] the first very large population trial, tested not only a scientific principle—whether dietary fat and cholesterol were related to risk of cardiovascular

disease (they were not)—but also whether the recommendations of health agencies were a rush to judgment (they were). The study had such a large political component that, as striking as the scientific outcome was—there was no effect of dietary total or saturated fat or cholesterol on cardiovascular disease—it couldn't be seen to fail. The results were buried for years until a statistician rediscovered them and finally had them published. This intertwining of the politics and science is a persistent pattern, and I try to tell both sides of the story and explain how they connect to each other.

My main principle, however, is that basic science comes first. I give preference to the demonstrations in nutritional and medical practice that are based in the fundamentals of biochemistry, of hard science. Big clinical trials have to be judged on their inherent strength, but, if they contradict basic science, the authors have an obligation to explain why. And science is continuous with common sense. It doesn't matter how many statistical tests you run: If the results violate common sense, it is unlikely to be science.

The poor research published by prestigious individuals and institutions suggests the nature of science itself has to be investigated. To do this, we will have to define scientific principles, explain how to read a scientific paper, and determine whether peer review has done its job. But first, in chapter 1, I give you the bottom line—the practical consequences of the science. The rest of the book will serve to justify these statements and recommendations.